Unusual Bottlenose Dolphin Mortality Event in the Swan Canning River Park, Western Australia.

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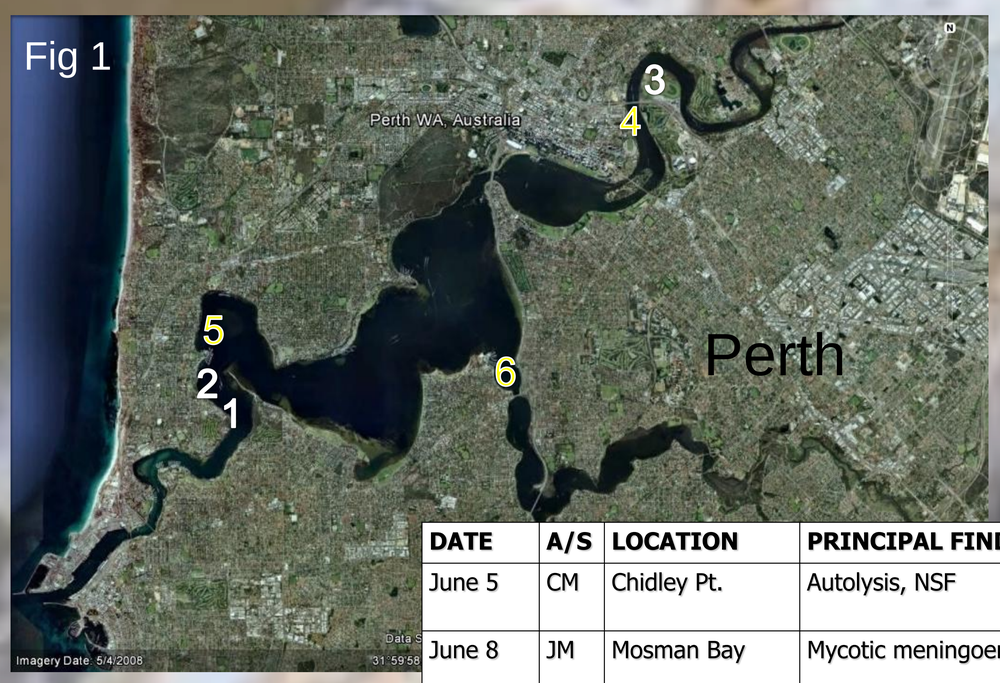
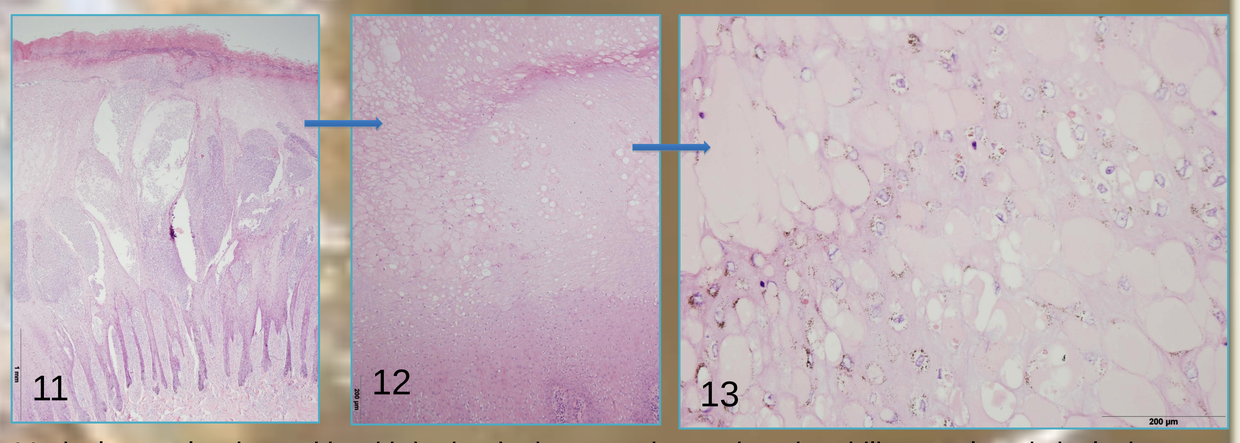
Introduction

Two clusters of mortality occurred in the Swan Canning River Park in 2009 resulting in 6 deaths from a resident community of 20 to 25 *Tursiops aduncus*. By comparison, only 6 deaths were recorded for the previous 7 years. Thus, 2009 was an anomalous year. The first cluster (1 to 3 in Fig. 1) occurred over 3 weeks in June (winter) while the second occurred over 5 weeks in September-October (spring).



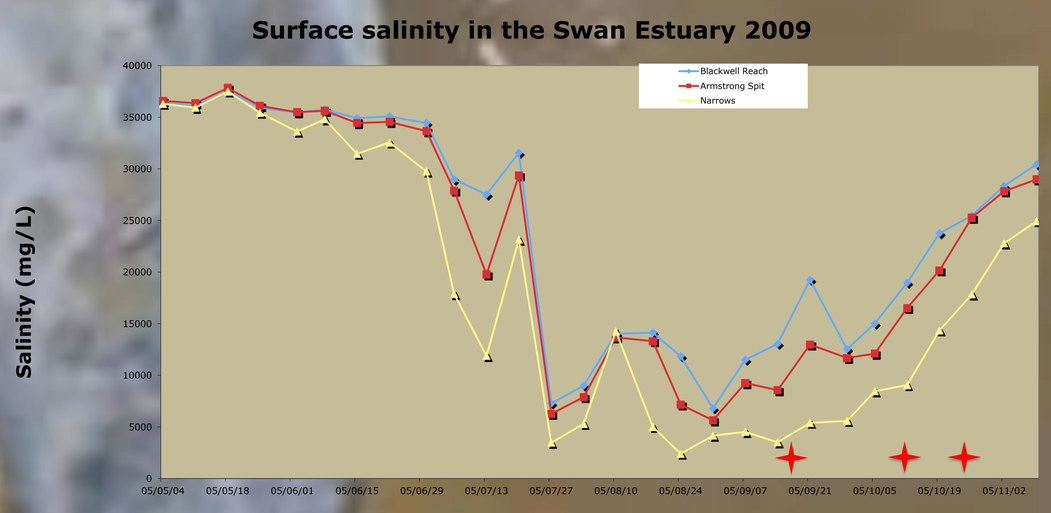
DATE	A/S	LOCATION	PRINCIPAL FINDINGS
June 5	CM	Chidley Pt.	Autolysis, NSF
June 8	JM	Mosman Bay	Mycotic meningoencephalitis
June 21	AF	Belmont	Chronic entanglement lesions, mycotic pneumonia, dermatitis
Sept 17	AF	Claisebrook Cove	Ulcerative dermatitis
Oct 9	AM	Freshwater Bay	Not necropsied
Oct 25	AF	Applecross Jetty	Ulcerative dermatitis



Vesiculopustular dermatitis with hydropic degeneration and eosinophilic cytoplasmic inclusions

Toxicology: Dieldrin levels were high in blubber (8.6 - 39 µg/kg lipid wt.). Sum PCBs expressed as µg PCB/g lipid weight ranged from 25.3 – 136.1 for the six dolphins. The reference value for marine mammals above which adverse effects on the immune response are thought to occur is 17 µg PCB/g blubber lipid[1]. Zinc levels were high (96 -160 µg/g wet weight) but the significance of this is unknown.

Water Salinity: Second cluster of deaths followed a rapid and significant decline in salinity following heavy spring rainfall.

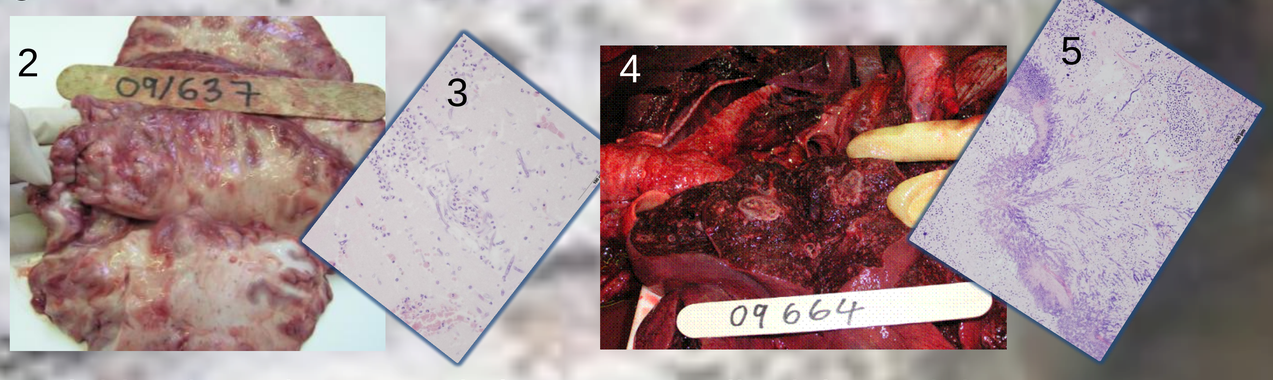


Results

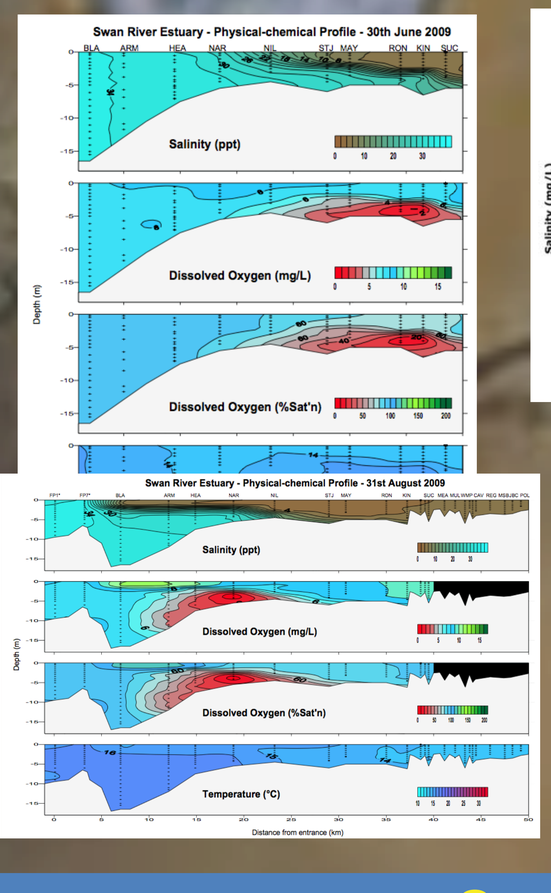
Pathology: The significant gross necropsy findings are summarized in the table above.

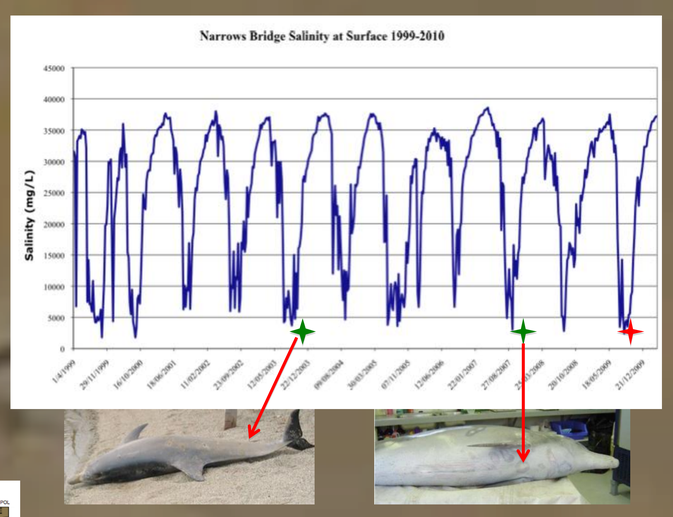
The dolphins in the first cluster were of mixed age classes. The June 8 and 21 animals died from opportunistic infections such as *Aspergillus* sp. pneumonia or encephalitis (Figs 2 - 5).

Characteristic dolphin morbillivirus lesions were not observed but CeMV antigen was detected in multiple tissues from the June 8 and 21 animals using standard immunohistochemistry methdology; PCR on the same tissues confirmed the presence of CeMV nucleoprotein gene.



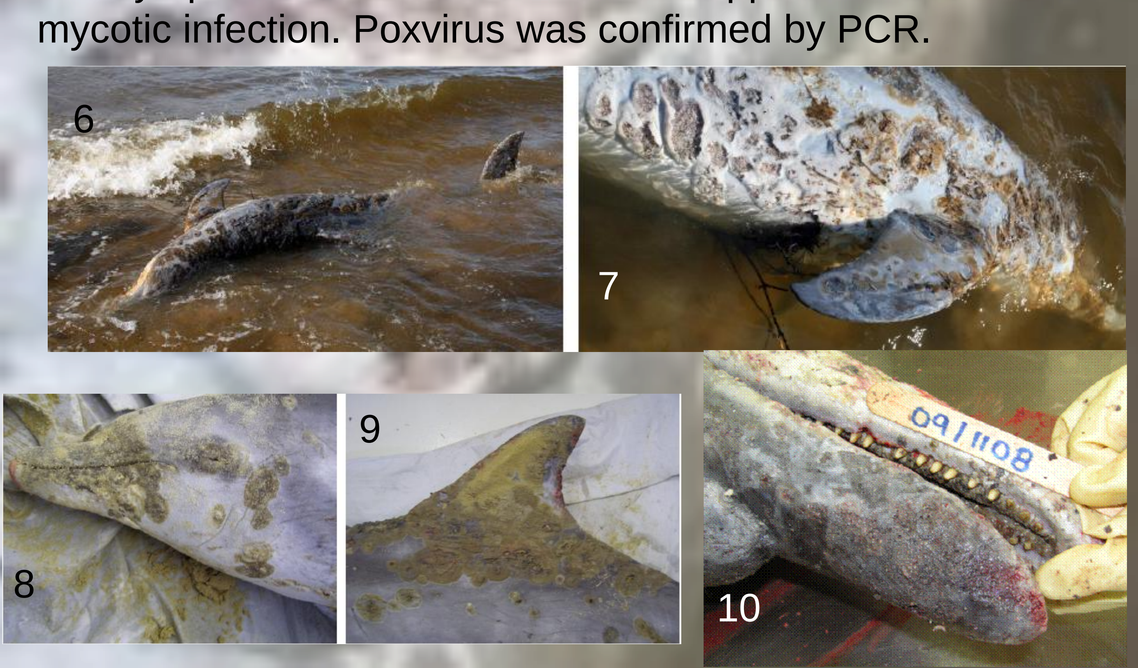
Dolphins from the second cluster were all adults and the most significant pathology was severe extensive ulcerative dermatitis (Background and Figs 6 – 13) associated with poxvirus intracytoplasmic inclusion bodies and opportunistic bacterial and mycotic infection. Poxvirus was confirmed by PCR.





Fluctuations in salinity occur annually. Analysis of the stranding records found 2 further examples of severe pox dermatitis in Oct ‘03 and Nov ’07 affecting adult dolphins.

Water quality data from the Swan River Trust: http://www.swanrivertrust.wa.gov.au/science/river/Content/plots.aspx



Conclusions

The June cluster of mortality appears to have been associated with an outbreak of cetacean morbillivirus. Mixed age classes suggests that this was a naïve population[2]. This is the first report of CeMV-related dolphin mortality from the Indian Ocean. The outbreak was coincident with a significant increase in strandings along the WA coast in 2009[3]. The Sept-Oct cluster followed the spring decline in water salinity and was associated with an extreme presentation of cetacean poxvirus causing extensive dermal ulceration exacerbated by osmotic stress. This is the first demonstration of poxvirus-associated mortality in adult dolphins[4]. The role of potentially immunosuppressive anthropogenic pollutants in either of these events is unknown.

[1]Jepson PD. *et al.* 2005. Relationships between PCBs and health status in UK-stranded harbour porpoises (*Phocoena phocoena*). *Environ. Toxicol. Chem.* 24: 238-248.
[2]Duignan PJ, *et al.* 1996. Morbillivirus infection in bottlenose dolphins: evidence for recurrent epizootics in the western Atlantic and Gulf of Mexico. Mar. Mammal Sci. 12: 499-515.
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[4]Van Bressem, M.F., *et al.* 2009. Epidemiological pattern of tattoo skin disease: a potential general health indicator for cetaceans. Dis. Aquat. Org. 85: 225-237.